fibrillation, and exercise-induced ventricular tachycardia may be a predictor of such an event. Beta-blockade with propranolol does not seem to protect against this, and specific antiarrhythmic treatment would probably have been more appropriate. We do not yet know, however, which of the several agents available for long-term prophylaxis of ventricular ectopic activity would best inhibit the short-cycle second ectopic beat that would appear to initiate the terminal arrhythmia.

The third patient was similar to the one reported by Pool *et al*,[4] with slow, bizarre complexes and asystole rather than ventricular fibrillation causing death. Liberthson and colleagues found that 28% of patients have terminal rhythms other than ventricular fibrillation.[5] Antiarrhythmic treatment is unlikely to be of any value in these cases.

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SHORT REPORTS

Screening for thyroid dysfunction in diabetics

The association between diabetes mellitus and primary hypothyroidism is well recognised, although the reported prevalence of hypothyroidism in diabetics varies from 0·2%[1] to 1·7%.[2] Routine biochemical screening of geriatric patients yields a prevalence of hypothyroidism of about 2% and is made more effective by additionally measuring serum thyrotrophin (TSH) concentrations. Thyroid function tests were performed and thyroid antibodies tested for in 255 consecutive diabetic outpatients who were not suspected of having thyroid disorders to determine the prevalence of unrecognised and subclinical hypothyroidism.

Patients, methods, and results

We recorded the details of the patients in advance from the case notes. Serum thyroxine, triiodothyronine uptake, free thyroxine index (FTI), and TSH were measured in duplicate, by radioimmunoassay, in postprandial venous blood samples. Normal values for our laboratory, determined from 180 healthy volunteers of similar ages, are concentrations of serum thyroxine 75-140 nmol/l (5·8-10·9 μg/100 ml), FTI 70-145, and TSH < 4·5 mU/l.

Clinical details and thyroid function in 255 diabetics. (Figures are numbers (%) of patients)

	Total	Group 1 (clinically hypothyroid)	Group 2 (raised TSH, FTI normal or low)	Group 3 (low FTI, normal or undetectable TSH)	Group 4 (normal FTI, normal or undetectable TSH)
No (%) of patients	255 (100)	7 (3)	77 (30)	25 (10)	146 (57)
Sex:					
Female	157 (62)	6 (86)	73 (95)	9 (36)	69 (47)
Male	98 (38)	1 (14)	4 (5)	16 (64)	77 (53)
Treatment:					
Diet	43 (17)	3 (43)	15 (19)	1 (4)	24 (16)
Tablets	102 (40)	3 (43)	34 (44)	5 (20)	60 (41)
Insulin	110 (43)	1 (14)	28 (36)	19 (76)	62 (42)
Age (years):					
<40	41 (16)	0	6 (8)	9 (36)	26 (18)
40-60	60 (24)	1 (14)	10 (13)	4 (16)	45 (31)
>60	154 (60)	6 (86)	61 (79)	12 (48)	75 (51)
Antibody:					
Present	67 (26)	5 (71)	26 (34)	5 (20)	31 (21)
Absent	188 (74)	2 (29)	51 (66)	20 (80)	115 (79)

TSH = Serum thyrotrophin.
FTI = Free thyroxine index.

Thyroid antibodies (microsomal and thyroglobulin) were detected by haemagglutination. Patients with a FTI < 60 and TSH concentration ≥10 mU/l were recalled, while those with a minor depression of FTI or slightly raised TSH concentrations were reassessed when they reattended the clinic. Statistical analysis was by χ^2 and Student's *t* tests.

Four distinct groups of patients emerged (table). There was a preponderance ($P<0·01$) in groups 1 and 2 of women patients and patients over 60 years. Thyroid antibodies were also more common ($P<0·05$) in groups 1 and 2 (three patients had been treated for hyperthyroidism) Over a quarter of group 2 have now been reviewed. Many have non-specific symptoms but no clinical hypothyroidism, although 10% had both raised TSH and antibodies. In group 3 there were significantly more men patients ($P<0·01$), patients under 40 ($P<0·05$), and patients receiving insulin treatment ($P<0·01$). There was no significant difference between the mean (±SE of mean) plasma glucose concentration in groups 1 and 2 (11·8 ± 0·7 mmol/l (212 ± 13 mg/100 ml)) and in group 3 (13·4 ± 1·5 mmol/l (241 ± 27 mg/100 ml)) or group 4 (11·5 ± 0·4 mmol/l (207 ± 7 mg/100 ml)). Three patients had been treated for hypothyroidism.

Comment

The prevalence of hypothyroidism (4%) was higher than that reported[1 2] owing mainly to the presence of unsuspected clinical hypothyroidism (2·7%). This may in part be attributed to the increased effectiveness of biochemical screening using FTI and TSH in determining which patients require detailed thyroid assessment. Most of these patients had mild clinical hypothyroidism and thyroid antibodies. The high prevalence of abnormal thyroid function tests may result from the prevalence (26%) of thyroid antibodies in diabetics, and the influence of poorly controlled diabetes on thyroid hormone concentrations.[3] Thirty per cent of diabetics had raised TSH (group 2) associated with a normal (17%) or low (13%) FTI but no clinical evidence of hypothyroidism. As in group 1, there was a preponderance of women over 60. The presence of thyroid antibodies or a raised TSH (subclinical hypothyroidism) may be associated with an increased risk of developing coronary artery disease, although the pathogenesis remains unclear.[1] There is an annual 2% incidence of overt hypothyroidism in subjects with both raised TSH and antibodies.[5] Thus 10% of our population may develop overt hypothyroidism and require annual thyroid function tests. A low FTI with normal or undetectable TSH (group 3) in young insulin-treated male patients may reflect the influence on thyroid function of insulin insufficiency or possible microvascular disease of the pituitary.

Screening thyroid function in diabetics, particularly elderly women, gives a yield (6%) greater than that found in the geriatric population. In addition to identifying undiagnosed clinically hypothyroid patients, it identifies an "at-risk" group with subclinical hypothyroidism. We feel that such screening is clinically and economically justifiable.

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